Management of Patients After Cardiac Arrest

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KEYWORDS

- Cardiac arrest • Postarrest care • Out-of-hospital cardiac arrest
- Targeted temperature management

KEY POINTS

- 12-Lead ECG is recommended in all survivors of cardiac arrest to evaluate for acute myocardial ischemia but is more prognostic at least 8 minutes after return of spontaneous circulation.
- Consider computed tomography chest, abdomen, and pelvis scan after resuscitation to identify underlying cardiac arrest causes and compression-induced traumatic injuries.
- Comatose survivors of cardiac arrest should undergo targeted temperature management, which can reasonably be actively maintaining normothermia at $\leq$37.7°C (often requiring cooling devices to achieve), although targeting mild hypothermia may be reasonable in select populations.
- Urgent coronary angiography for a non–ST-elevation myocardial infarction is not recommended for comatose survivors of cardiac arrest and instead can await neurologic improvement.

INTRODUCTION

Cardiac arrest, including both out-of-hospital cardiac arrest (OHCA) and in-hospital cardiac arrest (IHCA), is an individual medical emergency and a significant ongoing societal public health problem. Approximately 350,000 people in the United States experience nontraumatic OHCA each year.[1] Based on 2022 registry data, OHCA occurs in a predominantly male (62.7%), older population (median age, 65) and is frequently unwitnessed (51.3%) with asystole or pulseless electrical activity as the majority first arrest rhythm (74.7%).[2] In older adults the arrest cause is often due to cardiovascular and respiratory causes, while trauma and drug overdoses are more frequent in

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younger adults. Overall, only 9% to 10% of initial survivors of OHCA survive to hospital discharge, and only around 8% are discharged with a favorable neurologic outcome (Cerebral Performance Category 1 or 2, indicating at most moderate neurologic disability after discharge).[2,3] Although this indicates the poor prognosis of patients with OHCA, most OHCA mortality occurs in the out-of-hospital setting or in the emergency department, with only around 25% of OHCA patients surviving to hospital admission.[2] This underlines the importance of high-quality and vigilant medical care for survivors of OHCA once admitted to hospital. Survival to hospital discharge and survival with favorable neurologic outcome rates are significant higher, 30.7% and 27.3%, respectively, for bystander-witnessed OHCA with an initial shockable rhythm, namely ventricular tachycardia (VT)/ventricular fibrillation (VF).[4]

IHCA has substantially different epidemiology and outcomes. There are approximately 292,000 adult IHCAs in the United States per year, and arrests are more likely to be witnessed, with earlier initiation of high-quality cardiopulmonary resuscitation (CPR), and shorter time to return of spontaneous circulation (ROSC).[5,6] Patients with IHCA may have more underlying comorbidities and a higher rate of nonshockable rhythms, yet have a higher 30-day survival compared with patients with OHCA.[7] Survival to hospital discharge after IHCA in the United States in 2019 was 26.7%, with 18.0% of survivors having a good neurologic outcome.[8]

The most important factors governing the degree of organ injury and clinical course after resuscitation are the duration of ischemic time with absent or ineffective systemic circulation, and the clinical condition of the patient immediately before their arrest. There are now at least 80 published predictive models for outcomes postcardiac arrest and in the aggregate, IHCA or OHCA, initial arrest rhythm, age, low- or no-flow time (eg, duration and quality of CPR), and whether the arrest was witnessed emerge consistently as strong predictors.[9] The most well-validated models are the CAHP (Cardiac Arrest Hospital Prognosis) score,[10] the Out of Hospital Cardiac Arrest score[11] and the MIRACLE2 score[12] for OHCAs, and the GO-FAR (Good Outcome Following Attempted Resuscitation) score[13] for IHCA.

The core acute management goals for survivors of cardiac arrest are to provide organ support, sustain adequate hemodynamics, and evaluate the underlying cause of the cardiac arrest with the aim of survival with a good neurologic outcome. Achieving this goal requires management of all facets of the postcardiac arrest syndrome.[14] The authors review the current state of knowledge and clinical intensive care unit (ICU) practice recommendations after cardiac arrest, particularly focusing on important areas with specific unanswered questions. They note that there is a growing body of evidence for extracorporeal CPR,[15] but the management of patients receiving extracorporeal membrane oxygenation exceeds the scope of this review of generalized postarrest care.

DISCUSSION

Traumatic Injury

An underappreciated aspect of nontraumatic cardiac arrest pathologic condition is mechanical injury sustained during CPR, which may be neglected by physicians accustomed to working in medical- and cardiac-ICUs. Thoracic and extrathoracic injuries as a result of manual chest compressions are well documented, including easily imaginable rib and sternal fractures, but injuries to the heart, lungs, liver, gastrointestinal tract, and even to the vertebral bodies may occur. The adoption of mechanical CPR devices in recent years may be increasing the incidence of these injuries, specifically posterior rib fractures and heart and liver lesions, owing to device migration, excess mechanical force, and/or longer CPR duration.[16]

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These injuries may complicate several aspects of organ dysfunction after resuscitation. Pain from rib or sternal fractures may lead to atelectasis and poor secretion clearance after extubation and may contribute significantly to delirium, confounding neurologic assessment and prognostication. Extensive rib fractures may even cause flail chest requiring surgical stabilization with rib plating to improve respiratory mechanics.[17,18] Hypoxia and reduced lung compliance may be secondary to acute respiratory distress syndrome or aspiration, or to traumatic pulmonary contusions. Serial assessment of the shock state of the patient with postcardiac arrest should include consideration of hemorrhage (from lung or liver laceration), or tamponade (from pericardial laceration). Point-of-care ultrasound using a focused assessemnt with sonography for trauma (FAST) or extended-FAST traumatic injury protocol may be valuable diagnostically.[19] Mechanical injury to a broad range of anatomic structures has been identified after cardiac arrest, including gastric perforation after untrained bystander CPR.[20] As such, a "pan-scan" computed tomography (CT) scan immediately after resuscitation should be considered both to evaluate cardiac arrest causes and also to identify significant complicating injuries. In one cohort of 225 consecutive resuscitated patients with OHCA, rapid CT assessment of the head, chest, and abdomen/pelvis before ICU admission identified 42 critical findings potentially causing the cardiac arrest and 299 pathologic conditions regarded as a consequence of CPR and relevant for further treatment.[21] In this study, most critical findings were pulmonary emboli (40%) and tension pneumothorax (26%) with less frequent intracranial bleeding, aortic dissection, and pericardial tamponade, whereas the most frequent overall pathologic conditions were rib fractures (54%), aspiration (21%), and sternal fractures (17%).

Targeted Temperature Management

Postcardiac arrest brain injury results from ischemia during the no-flow or low-flow period, and from reperfusion injury after ROSC. Over the last 3 decades, a central component of reducing brain injury has been temperature management, now commonly termed targeted temperature management (TTM), which has included mild or moderate therapeutic hypothermia or "cooling" as well as active normothermia to prevent fever. The protective neurologic effects of hypothermia were identified from seemingly miraculous recoveries of avalanche or cold-water drowning victims after prolonged arrest, validated in animal studies, and hypothermia is commonly used to enable neuroprotection during cardiac surgery. Although in these scenarios cooling *precedes* low-flow or no-flow states, several randomized trials conducted from the mid-1990s onward have used therapeutic temperature management in comatose survivors *after* nontraumatic cardiac arrest, with significant variability in patient characteristics, cooling methods, temperature targets, and timing and duration of cooling (**Table 1**). The results have likewise been variable, and differences between society guidelines reflect this heterogeneity.

The Hypothermia after Cardiac Arrest (HACA) and Bernard trials in 2002 were the first major, albeit small, randomized trials to examine this question.[22,23] Collectively the trials recruited 352 comatose patients after OHCA with shockable rhythms, and randomized to initial therapeutic hypothermia (32°C–34°C for 24 hours and 33°C for 12 hours, respectively) followed by rewarming, or control arms without cooling. Both trials demonstrated improved neurologic outcomes in the induced hypothermia groups, with the HACA trial also demonstrating a significant mortality benefit (RR, 0.74; NNT, 7). Notably, temperature in the control arms was not well regulated, with median temperature of approximately 37.5°C and mean temperature of 37.4°C after

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Table 1
Target temperature management randomized trials

Trial (Year)	N	Eligibility[a]	Intervention	Control	Primary Outcome	Findings	Comments
HACA (2002)	273	VT/VF, witnessed OHCA with early CPR	32°C–34°C for 24 h, then passive rewarming	No specific target; Median ~37.5°C	Favorable neurologic outcome (CPC 1 or 2) at 6 mo	NNT 7 for mortality, NNT 6 for neurologic benefit	Restrictive eligibility of favorable patients. Control group hyperthermia
Bernard et al (2002)	77	VF OHCA	33°C for 12 h then active rewarming after 18 h	37°C for 12 h; mean 37.4°C	Survival to hospital discharge to either home or rehab facility	No mortality benefit; NNT 4 for neurologic benefit (adjusted OR 5.25)	Pseudo-randomized by day. Cooling started by EMS. Control group hyperthermia
TTM (2013)	939	OHCA of presumed cardiac cause[b]	33°C for 28 h, then rewarming by 0.5°C/h to 37°C	36°C for 28 h, then rewarming by 0.5°C/h to 37°C	All-cause mortality	No benefit for mortality or neurologic outcome	Mostly witnessed VT/VF with early CPR
HYPERION (2019)	581	Nonshockable rhythm OHCA or IHCA	33°C for 24 h, then rewarming by 0.25–0.5°C/h to 36.5–37.5°C, which was maintained for 24 h	36.5–37.5°C for 48 h	Favorable neurologic outcome (CPC 1 or 2) at 90 d	No mortality benefit; NNT 22 for neurologic benefit	Fragility index of 1. Excluded late CPR (begun >10 min)
TTM2 (2021)	1850	OHCA of presumed cardiac or unknown cause[b]	33°C for 28 h, then rewarming by 0.33°C/h to 37°C through 40 h, then 36.5°C −37.7°C through 72 h	<37.8°C for 28 h (37.5°C target when cooling devices used), then 36.5°C −37.7°C through 72 h	All-cause mortality at 6 mo	No difference in mortality or neurologic outcomes	46% in normothermia group received cooling device
HACA in-hospital (2023)	249	IHCA	32–34°C for 24 h, then rewarming at 0.25°C/h to 37.5°C until recovery	No protocol, target ≤37.5°C; Mean 37.0°C ± 0.9°C at 48 h	All-cause mortality after 180 d	No difference in mortality or neurologic outcomes	Early CPR, majority nonshockable

[a] All comatose survivors of cardiac arrest.
[b] Excluded unwitnessed arrest with asystole as initial rhythm.

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12 hours in the 2 trials, respectively, suggesting a significant degree of hyperthermia in the control groups.

The TTM trial (2013) recruited a larger (939) and broader cohort, including comatose survivors of OHCA of presumed cardiac cause and excluding unwitnessed arrest with asystole. TTM compared actively cooling patients to 33°C versus 36°C for 24 hours followed by gradual rewarming, and then temperature management to maintain less than 37.5°C for the following 72 hours.[24] There was no difference in mortality or neurologic disability between the groups. However, this still remained a relatively favorable group at baseline—overwhelmingly arrests were witnessed (89%) with a median basic life support initiation of 1 minute.

The HYPERION trial (2019) examined cooling to 33°C versus 37°C in 584 comatose patients with IHCA or OHCA with nonshockable rhythms, excluding patients with more than 10 minutes from collapse to initiation of CPR.[25] Patients in the hypothermia group had better neurologic outcomes compared with those in the normothermia group cerebral performance category (CPC) score at 90 days of 1 or 2 in 10.2% of hypothermia group vs 5.7% of normothermia group, NNT 22). However, troublingly, the confidence interval for the benefit of cooling was very wide—the study has been shown to have a fragility index of one.[26] In addition, cumulatively TTM was implemented for 56 to 64 hours in the hypothermia intervention group, whereas the control group received 48 hours of normothermia, and a significant number of patients had temperatures above 38°C after the period of TTM.

The TTM2 trial (2021) compared 1850 comatose patients with OHCA (74% initial shockable rhythm) that were cooled to 33°C versus active normothermia management ($\leq$37.7°C).[27] No differences in mortality or neurologic outcomes were observed, but there was a greater incidence of hemodynamically significant arrhythmias in the cooling group. Notably, the normothermia group received strict temperature control, with 46% receiving device-based cooling to maintain normothermia. These results were also demonstrated in a combined meta-analysis of TTM and TTM2.[28] For many clinicians this study has been decisive toward a practice of active avoidance of fever rather than therapeutic hypothermia in patients with OHCA. Nevertheless, clinical applicability of these trial data remains nuanced, particularly regarding the nonshockable initial rhythm and IHCA populations. The subsequent small HACA in-hospital trial (2022) compared cooling to 32°C to 24°C versus normothermia in 249 patients after IHCA (25% initial shockable rhythm) and also found no difference in mortality or in neurologic outcomes.

These trials also used different methods and durations of temperature intervention. Hypothermia, when therapeutically desired, should be instituted rapidly, typically with ice packs to areas of heat exchange and/or cooling devices. Limited animal studies suggest benefit in achieving faster cooling rates after arrest (eg, within 1 hour); however, prehospital cooling has not proven effective, and routine rapid infusion of cold intravenous fluid is not recommended.[29–31] At this time there are no clear data to recommend a specific therapeutic window in which hypothermia initiation is most effective. TTM duration has also been assessed in a randomized trial of 24 hours of TTM to less than 36°C, followed by either 12 or 48 hours of TTM less than 37°C, with no difference in mortality or neurologic outcome found.[32] Notably, the OHCA population in this trial was generally favorable with mainly shockable initial rhythms, witnessed arrests, and early CPR, and neither the control nor the treatment arms had tight temperature control in the latter phase.

Although the general takeaway of these trials appears to be that hypothermia can be discarded as an approach and may even be associated with harm,[33] there is an argument that certain groups of patients may benefit, particularly patients with significant

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ischemia-reperfusion brain injury. Whereas most studies focused on more prognostically favorable cardiac arrest patients (eg, shockable initial rhythms, witnessed arrest, and modest duration of CPR), the HYPERION trial suggested TTM benefit in patients with nonshockable rhythms, a population known to be associated with worse outcomes. Although this benefit was not seen in the nonshockable predominant HACA in-hospital trial, the patients in this trial had higher rates of witnessed arrest and early CPR, and a relatively short time to ROSC. Thus, duration of cerebral hypoperfusion and degree of brain injury may identify patients more likely to benefit from therapeutic hypothermia after cardiac arrest. A retrospective study stratifying 1111 patients by severity of postcardiac arrest illness found medium, but not high or low, severity risk scores showed both mortality and neurologic outcome benefits of cooling to 32°C to 34°C (compared with 35°C–36°C).[34] Conversely, a similar cohort study found TTM to 33°C was associated with survival benefit among patients with severe postcardiac arrest illness (based on organ failure scores, cerebral edema severity, and malignant electroencephalogram [EEG] patterns), but patients with mild to moderate severity illness appeared to benefit from TTM at 36°C.[35] A pooled analysis of published TTM data showed a strong negative association between rate of bystander CPR and TTM efficacy.[36] This suggests that populations at higher risk for significant neurologic injury (eg, less bystander CPR) may have the greatest benefit from therapeutic hypothermia. This could conceivably explain the outcome difference between predominantly VT/VF cardiac populations in TTM and TTM2 trials, and the nonshockable OHCA population in the HYPERION trial.

Although all this evidence may seem contradictory and bewildering, collectively current data suggest fever in the immediate postresuscitation period (36 hours or longer) is probably harmful for comatose survivors of cardiac arrest, and there is probably benefit in actively maintaining normothermia at $\leq$37.7°C (including invasive temperature monitoring and frequent use of cooling devices). However, in the authors' assessment, it is also reasonable to consider an initial 24 hours of mild to moderate hypothermia (33°C–36 °C) in patients with moderate or higher neurologic illness severity. Notably, these recommendations are inferred, not directly trial data supported, and contrast with society guidelines for TTM, which themselves disagree. The American Heart Association (AHA) recommends 32°C to 36°C for 24 hours[37]; the Neurocritical Care Society (NCC) does not provide a specific temperature management target,[38] and the American Academy of Neurology recommends 32°C to 34°C for 24 hours specifically for VT/VF, or 36°C for 24 hours,[39] whereas the joint European ResearchCouncil (ERC) and European Society of Intensive Care Medicine (ESICM) guidelines, which are the only ones at the time of writing this review that have been updated to the reflect the TTM2 trial, recommend continuous core temperature monitoring and actively maintaining normothermia at less than 37.7°C for at least 72 hours.[40] If hypothermia is implemented, shivering as well as metabolic, hematologic, immunologic, pharmacokinetic, and hemodynamic sequelae need to be carefully monitored and treated.[38]

Sedation

A core tenet of neuroprotection after cardiac arrest is reducing cerebral oxygen demand can reduce secondary injury from reperfusion, particularly that mediated by neuronal excitotoxicity. Clinical trials have largely focused on TTM to reduce cerebral oxygen demand in the critical postarrest period. Deep sedation has also been a standard part of TTM protocols though. This conflicts with current prevailing ICU practice of minimizing sedation in nonarrest intubated populations.[41,42] There are no human studies directly addressing sedation for postcardiac arrest neuroprotection, although

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this is an area of ongoing and active research.[43] In postcardiac arrest mice undergoing therapeutic hypothermia, sedation with propofol or dexmedetomidine had better outcomes in terms of survival, neurologic function, EEG findings, cerebral blood flow, and neuronal cell death on histology compared with no sedation.[44]

Indirect evidence, perhaps surprisingly, comes from studies of TTM, where 2 of the 3 major trials that showed a positive effect of postresuscitative cooling had also significant differences in sedation strategy. The HACA trial similarly mandated sedation, analgesia, and neuromuscular blockade (NMB) in both intervention and control groups for 32 hours, whereas the Bernard trial only maintained sedation and NMB in the intervention group (after initial treatment in both groups), and the HYPERION trial specifically targeted deep sedation in the intervention group and mild sedation for the initial 12 hours in the control group. Conversely, in the major negative trials on TTM, sedation strategies were kept tightly similar between groups. The TTM protocol mandated sedation in both arms (without a specified target, although sites were instructed to treat the groups similarly), and in TTM2 an identical deep sedation target was mandated in both control and intervention groups.[24,27] The confounding of important differences in sedation in positive therapeutic hypothermia studies and its absence in negative studies is suggestive, and there may be fruitful research in this direction in the near future. For patients undergoing therapeutic hypothermia, all trials and guidelines included sedation (and paralysis as needed to prevent shivering) and should be used. For patients undergoing active normothermia, there is no direct evidence to support routine sedation beyond the standard of ICU care. Instead, sedation could limit neurologic assessment and delay time to awakening. This concern is corroborated by evidence suggesting that use of shorter-acting sedation with propofol is associated with earlier awakening after cardiac arrest compared with a midazolam-fentanyl protocol.[45] However, a post hoc analysis of TTM trial time to awakening found delayed awakening is relatively common but could not be attributed to differences in sedation.[46]

Acknowledging the lack of definitive data, the recently updated ERC/ESICM guidelines highlight the TTM2 trial sedation protocol and explicitly make no recommendation on sedation use or duration.[40] Based on current data, the authors' clinical practice uses routine short-acting sedation in the initial postcardiac arrest period.

Neuroprognostication

Assessment of neurologic function in the comatose survivor of cardiac arrest focuses on 2 main domains: identification of alternative causes to coma and, otherwise, attempting to predict the likelihood, timing, and degree of functional neurologic recovery. The AHA adult postcardiac arrest care algorithm recommends obtaining a brain CT and an EEG in all comatose survivors of cardiac arrest as part of the evaluation for rapidly reversible causes to coma, such as intracranial hemorrhage or nonconvulsive status epilepticus, respectively.[47]

There are a few clear best practices for neuroprognostic assessment, and many areas of ongoing research. Neurologic assessment should wait at least 72 hours following rewarming and should be extended for a period of prolonged observation in patients with an indeterminant prognosis, if consistent with established goals of care.[48] This observation period is crucial, as about half of OHCA survivors will remain comatose 72 hours after ROSC/rewarming,[49] and early withdrawal of life-sustaining therapies may be a cause of excess mortality.[50] If any purposeful movement is noted, the neurologic outcome is no longer indeterminant, as the patient is no longer in a coma, recognizing that the degree of persistent functional debility will likely still remain unknown. Reliable predictors of neurologic outcome in survivors of cardiac arrest who

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remain comatose per NCC guidelines are as follows: bilateral absence of pupillary light response greater than 72 hours from ROSC and bilateral absence of N20 response on somatosensory evoked potential (SSEP) testing, whereas CT or MRI of the brain greater than 48 hours from ROSC and electroencephalography greater than 72 hours from ROSC are moderately reliable predictors.[48] A range of biochemical neuroprognostication predictors continue to be assessed and may assist the neurologic evaluation, including neuron-specific enolase (NSE), plasma neurofilament light,[51] and p-tau.[52] Postarrest myoclonus is associated with poor outcome but not invariably or reliably.[53] However, postarrest seizure is common (occurring in 29% of patients in the TTM trial) and is predictive of poor outcome.[54] Alternatively, a withdrawal or localization motor response to pain immediately or at 72 to 96 hours after ROSC, normal NSE 24 to 72 hours after ROSC, short-latency SSEP wave amplitude greater than 4 μV, or continuous background without discharges on EEG within 72 hours from ROSC and absent diffusion restriction on MRI on days 2 to 7 after ROSC predicted good neurologic outcome with 80% specificity and low sensitivity.[55] Overall, the AHA neuroprognostication algorithm recommends a multimodal and multidisciplinary assessment, including brain MRI, NSE, SSEP, EEG, and clinical examination, which should be synthesized into a collective assessment at least 72 hours after achieving normothermia.[47]

Hemodynamics

The cardiovascular goals in the postresuscitation period are maintaining end-organ perfusion, particularly cerebral perfusion, and for certain patients, restoring coronary perfusion. The ERC and AHA recommend frequent noninvasive blood pressure monitoring or the use of arterial cannulation, and the ERC suggests cardiac output monitoring may be appropriate for some patients, without specifying a method. Clinical perfusion examination and echocardiography may be adequate for many patients. Echocardiography after cardiac arrest can also aid in determining arrest cause and guide management, including detecting myocardial stunning, regional wall motion abnormalities, structural abnormalities, effusions, or cardiomyopathy. Invasive hemodynamic monitoring with a pulmonary artery catheter may be of assistance in complex, unclear, or severe shock states. Other methods of estimating cardiac output may be useful in select scenarios, including trauma resuscitation.[56]

Society guidance for hemodynamic management in this period is not wildly divergent from general intensive care management. The AHA guidelines recommend mean arterial pressure (MAP) greater than 65 mm Hg and systolic blood pressure (SBP) greater than 90 mm Hg, using vasopressors, inotropes, or crystalloid as appropriate.[37] The ERC and ESICM recommend MAP at least 65 mm Hg, SBP greater than 100 mm Hg, and using lactic acid levels (normal or decreasing) and urine output (>0.5 cc/kg) to guide hemodynamic resuscitation. Notably, shock may be primarily low output from myocardial dysfunction (whether from cardiac stunning or ongoing ischemia), vasodilatation (owing to systemic inflammatory response syndrome-like features of postresuscitation syndrome), or a mixture of states. As such, vasopressors might have variable, contrasting, or paradoxical effects on cerebral perfusion and myocardial work depending on the shock state, vasopressor used, achieved blood pressure, and severity of cerebral and coronary vascular disease. A core goal is to maintain *cerebral* perfusion, and there is evidence suggesting that most patients post-cardiac arrest have impaired cerebral blood flow autoregulation and may require a higher MAP to maintain perfusion.[57] In addition, therapeutic hypothermia contributes to elevated systemic vascular resistance, which may have hemodynamic consequences.[58,59]

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Observational data consistently show that hypotension is associated with worse outcomes,[60,61] but have been inconsistent in determining an optimal higher blood pressure target or method of resuscitation after cardiac arrest.[62–65] The randomized, double-blinded blood pressure and oxygenation targets in post resuscitation care (BOX) trial compared MAP targets of 63 mm Hg and a modestly elevated 77 mm Hg in comatose survivors of OHCA of presumed cardiac cause, finding no difference in death or neurologic outcome, but that the higher MAP group required higher doses of vasopressor.[66] Notably, a small, but supportive, BOX substudy found cerebral biochemical patterns were predictive of poor outcome but not modified by higher MAP.[67] Alternatively, perhaps MAP is not the right target parameter in postcardiac arrest patients, as cerebral hypoperfusion can occur despite systemic normotension owing to low stroke volume from cardiogenic shock or increased intracranial pressure impairing cerebral blood flow. Limited observational data suggest that cardiac output after resuscitation is not associated with mortality,[68] but intracranial pressure and brain tissue oxygenation support cerebral blood flow optimization requiring individualized MAP targets.[69–71] Overall, at this time clinical data and guidelines do not provide clear guidance on hemodynamic targets outside of avoiding hypotension, generally by targeting MAP greater than 65 mm Hg.

Coronary Evaluation

Consideration for urgent coronary angiography is a core part of postarrest care, because coronary artery disease is a common cause of sudden cardiac death, can contribute to ongoing myocardial and electrical dysfunction, and may be treatable. The AHA's adult postcardiac arrest care algorithm includes obtaining a 12-lead electrocardiogram (ECG) in the initial stabilization phase after ROSC,[47] and OHCA survivors who are not comatose should be urgently assessed for acute myocardial ischemia. The urgency and therapeutic benefit of coronary angiography in *comatose* survivors of cardiac arrest have been more controversial.

Initial observational data identified a high incidence of significant coronary lesions in patients with postcardiac arrest, including 70% of all patients, 96% of patients with ST-elevation myocardial infarction (STEMI) ECG pattern, and 58% of non-STEMI patients.[72] Similarly in observational data, successful coronary angioplasty was associated with hospital survival.[73] However, multiple impactful randomized trials of comatose survivors of cardiac arrest with non-STEMI patterns on ECG have all demonstrated that early invasive angiography is no better than delaying angiography until after neurologic evaluation and recovery.[74–76] Meta-analysis similarly found no benefit of early coronary angiography in the combined post-OHCA non-STEMI population.[76,77] Crucially, each of these trials excluded patients with cardiogenic shock, recurrent life-threatening arrhythmias, or ongoing ischemia, and as such, each of these generally remain indications for urgent or emergent coronary angiography. However, recent observational data suggest that postarrest hemodynamic instability does not identify patients with acute coronary occlusion nor is predictive of fatal outcomes.[78]

In addition, these trials all excluded patients with a post-OHCA STEMI pattern on ECG, as STEMI remained an indication for emergent coronary angiography. However, the specificity of STE on the post-ROSC ECG is only approximately 85%,[79] as periods of hypoperfusion and reperfusion and the vascular effects of epinephrine may result in apparently pathologic ECG changes that resolve with time. In a study of 370 patients with postcardiac arrest whose post-ROSC ECG met STEMI criteria and who were then taken for angiography, 18.5% with ECG obtained within 7 minutes of ROSC had no culprit coronary lesion identified, whereas only 7.2% had false-positive STE on ECG if it was recorded 8 or more minutes after ROSC.[80] Regardless, the Society for

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Cardiovascular Angiography and Interventions (SCAI) guidelines on OHCA advocates for using assessment of multiple favorable and unfavorable clinical factors to distinguish between comatose survivors of cardiac arrest who should definitely be treated with an early invasive angiography strategy versus a deferral strategy, even for STEMI or cardiogenic shock.[81] The SCAI guidelines endorse a deferral strategy for all comatose survivors of cardiac arrest with non-STEMI.

In summary, patients with OHCA whose post-ROSC ECG, recorded 8 or more minutes after ROSC, meet STEMI criteria should usually receive urgent coronary angiography, while delaying angiography until neurologic recovery in a comatose survivor of a cardiac arrest is generally acceptable when there is a non-STEMI post-ROSC ECG. Urgent coronary angiography should still be considered for all patients with cardiogenic shock potentially owing to coronary ischemia, recurrent life-threatening arrhythmia (particularly VT or VF), or recurrent or ongoing ischemia (such as a rapidly rising cardiac troponin, dynamic ECG changes concerning for ischemia), although the benefits are less certain.

Ventilator Management

Postarrest optimization of oxygenation and ventilation is physiologically important as both tissue hypoxia and hyperoxia, with resultant harmful oxygen free radicals, may be deleterious, and carbon dioxide is an important regulatory of vascular tone. The randomized BOX trial found no difference in death or severe neurologic impairment with a restrictive or liberal oxygenation strategy.[82] However, the similar EXACT trial found that a conservative oxygen target was associated with worse outcome than a higher oxygen target in the prehospital setting.[83] Rationalizing this discrepancy, relative hypoxia may be more poorly tolerated in the immediate post-ROSC phase in the prehospital setting. Alternatively, in the BOX trial oxygen was increased if saturation dropped to less than 93% on peripheral pulse oximetry, whereas the conservative arm oxygen target of the EXACT trial was 90% to 94%. Given that guidelines have not been updated to include these newer trials, maintaining a pulse oximetry saturation greater than 93% appears appropriate. Regarding ventilation, the AHA algorithm recommends $Paco_2$ of 35 to 45 mm Hg, whereas the ERC/ESICM guidelines acknowledge a lack of data and recommend normocapnia.

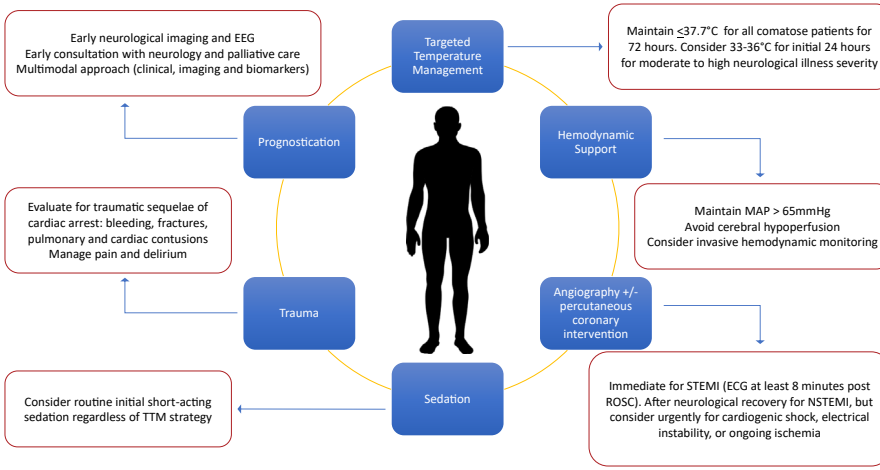
Early neurological imaging and EEG
Early consultation with neurology and palliative care
Multimodal approach (clinical, imaging and biomarkers)
Targeted Temperature Management
Maintain ≤37.7°C for all comatose patients for 72 hours. Consider 33-36°C for initial 24 hours for moderate to high neurological illness severity
Prognostication
Hemodynamic Support
Maintain MAP > 65mmHg
Avoid cerebral hypoperfusion
Consider invasive hemodynamic monitoring
Evaluate for traumatic sequelae of cardiac arrest: bleeding, fractures, pulmonary and cardiac contusions
Manage pain and delirium
Trauma
Angiography +/- percutaneous coronary intervention
Immediate for STEMI (ECG at least 8 minutes post ROSC). After neurological recovery for NSTEMI, but consider urgently for cardiogenic shock, electrical instability, or ongoing ischemia
Sedation
Consider routine initial short-acting sedation regardless of TTM strategy

Fig. 1. Postcardiac arrest care improvement. (Human figure from Wikimedia Commons. https://commons.wikimedia.org/wiki/File:SVG_Human_Silhouette.svg under CC-BY-SA-3.0).

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End-of-Life Care

Most deaths of initially comatose survivors of cardiac arrest are attributable to post-arrest brain injury and active withdrawal of life-sustaining treatment based on a predicted poor neurologic outcome. Comprehensive and multidisciplinary, including neurology and palliative care services, family counseling early in the clinical course is often helpful to begin addressing goals of care for a range of different potential outcomes. In addition, the organ donation team, distinct from the clinical ICU team, should be contacted per local and/or institutional guidelines before withdrawal of life-sustaining treatment.

SUMMARY

Although cardiac arrest remains a significant cause of morbidity and mortality, myriad dramatic advances, particularly in critical care medicine and cardiology, now enable potential survival with good neurologic outcome. Specifically, societal education on CPR algorithms, advanced diagnostic imaging, percutaneous coronary angiography for selected patients, hemodynamic monitoring and support platforms, TTM, neuroprognostication strategies, and contemporary ICU clinical practices have all contributed to postcardiac arrest care improvement (Fig. 1). Possibly the most difficult yet important clinical question is neuroprognostication, which remains fraught with uncertainty despite many advances in this domain.

For the practical-minded intensivist, these patients may be thought of as being in a highly precarious position, but also in a position to truly benefit from high-level critical care medicine. Although at present many core clinical questions remain unsettled, we look forward to further advances in the state of knowledge in the coming years.

CLINICS CARE PPOINTS

- Comprehensive and multi-disciplinary care is essential for managing patients after cardiac arrest.
- Post-arrest neuroprognostication of a comatose survivor of cardiac arrest should include a multimodal and multidisciplinary assessment at least 72 hours after achieving normothermia.
- ST elevations on the post arrest ECG are less likely to be falsely positive for culprit coronary lesions if the ECG is obtained at least 8 minutes after return of spontaneous circulation.

DISCLOSURES

The authors have no conflicts of interest relevant to this topic to disclose.

DEDICATION

Dedicated to the memory of Dr Ainsley Pratt, who shone brightly as a person and as a physician, and whose devotion to the care of her patients and colleagues continues to inspire all who knew her.

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